

Penicillin-Binding Protein 2x in Streptococcus pneumoniae Isolated from Tehran Hospitals

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ABSTRACT

Background and objectives: Globally, millions die each year from infections caused by *Streptococcus pneumoniae*. The effective treatment of pneumococcal infections has been compromised by the emergence of strains resistant to currently available antibiotics. In resistant strains there is a reduction in the capacity to bind to antibiotics in penicillin binding proteins. The purpose of this study was to determine the rates of antibiotics resistance in clinical strains of *Streptococcus pneumoniae* and analyse occurred mutations in pbp2x.

Materials and methods: 58 pneumococcal isolates were collected from patients with pneumococcal infections. The drug susceptibility patterns of the strains were tested by the disc diffusion method for penicillin G, cefotaxime, erythromycin, tetracycline, and trimethoprim-sulfamethoxazole. MICs of penicillin and cefotaxime determined by broth microdilution. *lytA* and *pbp2x* genes were amplified by PCR. The nucleotide sequences were determined by direct sequencing.

Results: From all 58 *Streptococcus pneumoniae* strains, 24 (41.3%) were penicillin intermediate and 14 isolates (24.1%) were penicillin resistant ($MIC \geq 2 \mu g/ml$). 28 (48.2%) were trimethoprim-sulfamethoxazole resistant, 9 (15.5%) were erythromycin resistant and 10 (17.2%) were tetracycline resistant. 8 isolates (13.8%) were cefotaxime resistant. Most of the strains resistant to penicillin and cefotaxime showed additional resistance to one or several other drugs. All of the cefotaxime resistant isolates had alterations in PBP2x.

Conclusion: Prevalence of beta-lactam resistant strains revealed a crisis in treatment of pneumococcal infections. We showed that alterations in the conserved motifs in PBP2x are associated with cefotaxime resistant in *Streptococcus pneumoniae*.

Key words: *Streptococcus pneumoniae*, PBP2X, beta-lactam Resistance